Figure 5: Presence of the inhibitory activity in protein fractions of HDL. HS was fractionated by high density centrifugation and the inhibitory activity of HDL and serum protein fractions was analyzed. Isolated HDL were further subjected to either delipidation (delipidated HDL) or proteolytic digestion with proteinase K (Proteinase K treated HDL). The inhibitory activity of fractions was compared to HS (whole serum). The final protein concentration for whole serum and serum proteins was 7 mg/ml (stippled columns), 3.5 mg/ml (hatched columns), and 0.7 mg/ml (white columns). The final protein concentration for HDL and delipidated HDL was 0.2 mg/ml (stippled columns), 0.1 mg/ml (hatched columns), and 0.02 mg/ml (white columns). The amount of proteinase K-treated HDL was estimated according to the protein concentration before proteolysis and was similar to untreated HDL. Results represent the percentage of IL-1β (Fig. 5B) or TNFα (Fig. 5A) production in the absence of inhibitor (mean .+-.SD, n=3).

Please replace the paragraph beginning at line 23 on page 11 with the following amended paragraph:

[0061] FIG. 6: Analysis of HDL binding to cells. (A) Inhibition of T cell-signalling by binding of HDL to membranes of stimulated HUT-78 cells; either membranes of stimulated HUT-78 cells (white columns), THP-1 cells (hatched columns), or both (stippled columns) were preincubated in the absence (-) or presence of FCS (10%), HS (10%) or HDL (0.32 mg/ml protein) for 45 minutes on ice; after washing, treated (hatched and stippled columns) and untreated (white columns) THP-1 cells were cultured in the presence of treated (white and stippled columns) or untreated (hatched columns) membranes of stimulated HUT-78 cells; TNFα and IL-1β production was

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measured in 48 hours-culture supernatants. Results are expressed as percentage considering the production measured in the absence of inhibitor as 100%, mean .+-.SD, n=6. (B-F) Binding of unconjugated FITC and FITC-HDL (0.1 mg/ml) was assessed by flow cytometry on THP-1 cells (B), isolated human monocytes (C), unstimulated HUT-78 cells (D) and stimulated HUT-78 cells (E). FITC was used as a negative control. (F) Binding of FITC-HDL (10 µg/ml) to stimulated HUT-78 cells in the presence or absence of purified anti-apo-A-I antibodies (100 µg/ml) (ATCC, Manassas, Va.; catalogue number HB-9570).

Please replace the paragraph beginning at line 16 on page 13 with the following amended paragraph:

Figure 9: Apo A-I inhibits TNFα and IL-1β in PBMC stimulated [bu] by either PHA or Tetanus Toxoid (TT). PBMC 4 X 10^5 cells/200 µl/well were stimulated by 1 µg/ml PHA (A and B) or by 10 µg/ml TT (C and D) in the presence of the indicated doses of apo A-I and HDL.

IN THE CLAIMS:

Please amend claims 9, 10, 15, and 16, as follows:

9. (Amended) An apo-A-1 fragment T-cell activation inhibitor-like polypeptide fragment produced by a process comprising culturing a eukaryotic cell comprising a vector comprising a nucleic acid molecule consisting essentially of a nucleotide sequence selected from:

(a) the nucleotide sequence as set forth in residues 73 to 601 in SEQ ID NO:1;

(b) a nucleotide sequence encoding the polypeptide as set forth in residues 25 to 194 in SEQ ID NO:2;

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